

Acid-Base Interpretation: A Practical Approach

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Acid-base disorders are prevalent in critically ill patients, and a systematic approach is essential for evaluation. The first step is to determine the primary process based on a patient's pH, partial pressure of carbon dioxide, and bicarbonate measurements. After this is complete, the next step is to evaluate for respiratory or metabolic compensation. Deviations from expected compensation may indicate additional acid-base processes. For metabolic acidosis, anion gap calculation distinguishes between anion gap metabolic acidosis and non-anion gap metabolic acidosis. The evaluation for anion gap metabolic acidosis includes calculating the osmolal gap and conducting a gap-gap analysis. Evaluating non-anion gap metabolic acidosis involves urine anion gap calculation. These analyses identify potential etiologies and additional acid-base disturbances. Metabolic alkalosis assessment begins with measuring urine chloride levels to determine whether the process is a result of chloride depletion. Respiratory acidosis, caused by hypoventilation, often results from chronic lung disease or neuromuscular dysfunction. Respiratory alkalosis, due to hyperventilation, is common in sepsis, chronic liver disease, and acute pulmonary embolism.

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Acid-base disorders are common in patients who are critically ill.¹ A systematic approach to evaluating these disorders provides vital diagnostic information and guides timely treatment.²⁻⁵ Acidosis leads to acidemia (pH less than 7.38)⁶ and is typically due to one of three factors: (1) accumulation of acids (eg, salicylic acid, keto acids, lactic acid) or loss of alkali (as in diarrhea or proximal renal tubular acidosis); (2) decreased renal excretion of acid (as in kidney failure or distal renal tubular acidosis); or (3) accumulation of carbon dioxide in the body. Alkalosis is a process that leads to alkalemia (pH greater than 7.42) and is primarily caused by excessive carbon dioxide exhalation (respiratory alkalosis), the accumulation of alkali (sodium bicarbonate [HCO₃]⁻ administration), or loss of acid (as with gastrointestinal loss from vomiting and diarrhea).⁷

APPROACH

The physiologic approach to acid-base analysis is based on the concept of the carbonic acid-HCO₃⁻ buffer system as a center of homeostatic control of blood pH.^{2-4,8,9} In addition to assessment of vital signs and fluid status, blood gas measurement and basic chemistry panel are required. Venous blood gas often is measured initially instead of arterial blood gas because it is easier to obtain and may reveal metabolic changes earlier in cases of circulatory insufficiency.¹⁰ An arterial blood gas measurement is preferred when assessing a patient's oxygenation status.

Primary Process

The first step is to determine serum pH level (normal serum pH is 7.38-7.42).² The next step is to identify the primary

disturbance as metabolic or respiratory by comparing pH and arterial PaCO₂ or estimated PaCO₂ from venous blood (PaCO₂ = PvCO₂ - 6 mm Hg)¹¹ with normal values (Table 1^{2,5,6,11-14}).

If pH and PaCO₂ levels are both decreased, the primary process is metabolic acidosis. If the pH level is decreased and the PaCO₂ level is increased, it is respiratory acidosis. If the pH and PaCO₂ levels are both increased, it is metabolic alkalosis. If the pH level is increased but PaCO₂ level is decreased, it is respiratory alkalosis.^{3,5} Figure 1 summarizes the initial evaluation of acid-base disorders.^{2,5,15}

Compensation

The second step is to evaluate whether metabolic or respiratory compensation is appropriate using a specific formula to calculate expected PaCO₂ compensation for metabolic disturbances

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SORT: KEY RECOMMENDATIONS FOR PRACTICE

Clinical recommendation	Evidence rating	Comments
Using a systematic approach to evaluating acid-base disorders provides vital diagnostic information and guides timely treatment in critically ill patients. ²⁻⁵	C	Reviews based on usual practice, consensus
To determine the primary process of an acid-base disorder, the pH and P_{aCO_2} on blood gas should be evaluated, noting that abnormal values increasing or decreasing in the same direction indicate a metabolic process and those shifting in opposite directions indicate a respiratory process. ^{3,5}	C	Review based on usual practice, consensus
When evaluating acid-base disorders, expected patterns of P_{aCO_2} compensation for metabolic disturbances and HCO_3^- compensation for respiratory disturbances should be reviewed to determine if an additional acid-base disorder is present. ^{3,5}	C	Usual practice, consensus
HCO_3^- = bicarbonate. A = consistent, good-quality patient-oriented evidence; B = inconsistent or limited-quality patient-oriented evidence; C = consensus, disease-oriented evidence, usual practice, expert opinion, or case series. For information about the SORT evidence rating system, go to https://www.aafp.org/afpsort .		

TABLE 1

Normal Values for Acid-Base Analysis

Measurement	Normal value	Reference range
pH	7.4	7.38-7.42
P_{aCO_2}	40 mm Hg (5.32 kPa)	38-42 mm Hg (5.05-5.59 kPa)
P_{vCO_2}	$P_{vCO_2} = P_{aCO_2} + 6$ mm Hg	≤ 48 mm Hg (6.38 kPa)
HCO_3^-	24 mEq/L (24 mmol/L)	22-26 mEq/L (22-26 mmol/L)
Anion gap = $Na - (Cl + HCO_3^-)$	≤ 12 mEq/L (12 mmol/L)	8-14 mEq/L (8-14 mmol/L)
Calculated osmolality = $2 \times Na + \text{glucose}/18 + \text{BUN}/2.8$	285 mOsm/kg (285 mmol/kg)	280-295 mOsm/L (280 mmol/kg)
Osmolal gap = measured – calculated osmolality	< 10 mOsm/L (10 mmol/kg)	10 ± 6 mOsm/L (10 ± 6 mmol/kg)
Urine anion gap = urine Na + urine K – urine Cl	< 0 mEq/L (0 mmol/L)	0 ± 10 mEq/L (0 ± 10 mmol/L)

BUN = serum urea nitrogen; Cl = chloride; HCO_3^- = bicarbonate; K = potassium; Na = sodium.

Information from references 2, 5, 6, and 11-14.

or expected HCO_3^- compensation for respiratory disturbances (Table 2^{3,5}). If measured values deviate significantly from calculated expected values, a mixed acid-base disturbance is present (eg, an uncompensated acidosis or alkalosis, or a second acid-base disturbance).^{3,5}

It is important to note that critically ill patients often present with, or develop, mixed acid-base disturbances, particularly when serum HCO_3^- and P_{aCO_2} levels deviate in opposite directions from their normal values.⁴ In complex cases or when

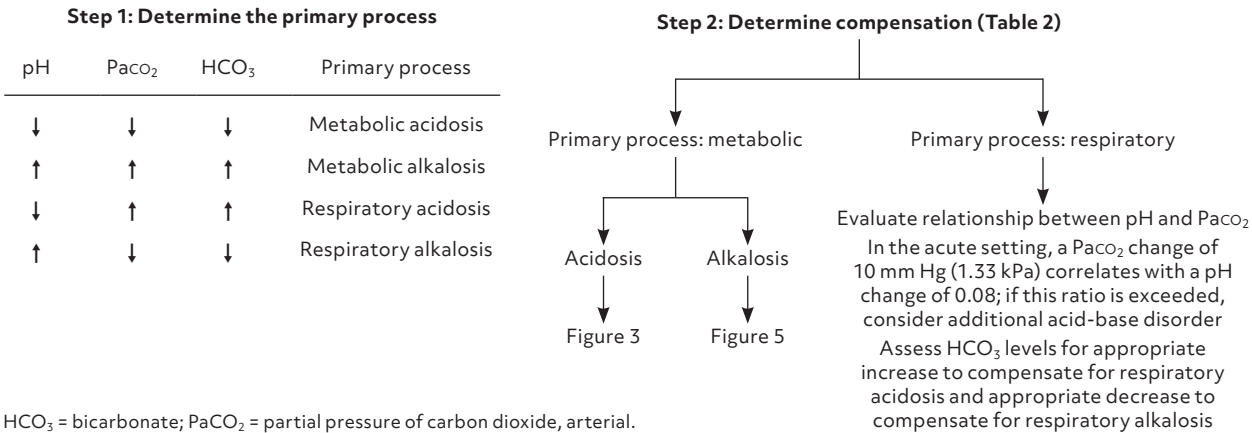
treatment does not yield expected improvements, consultation with an intensive care specialist or nephrologist is advisable.

CASE 1

A 48-year-old patient with type 2 diabetes is admitted for acute confusion and diarrhea. The patient is afebrile and normotensive. Laboratory results are as follows:

- Arterial blood gas: pH = 7.30, P_{aCO_2} = 25 mm Hg (3.33 kPa), P_{aO_2} = 85 mm Hg (11.30 kPa) on room air

FIGURE 1



Initial evaluation of acid-base disorders. Step 1 is to determine the primary process. After the primary process is identified, step 2 is to examine whether compensation is appropriate. If not, an additional acid-base process exists. Then, algorithms for individual acid-base disorders should be followed.

Information from references 2, 5, and 15.

- Serum: sodium = 148 mEq/L (148 mmol/L), potassium = 3.2 mEq/L (3.2 mmol/L), chloride = 104 mEq/L (104 mmol/L), HCO₃ = 10 mEq/L (10 mmol/L), urea nitrogen = 35 mg/dL (12.49 mmol/L), creatinine = 1.8 mg/dL (159.12 μmol/L), glucose = 360 mg/dL (19.98 mmol/L), osmolality = 330 mOsm/kg (330 mmol/kg)

Comparison of the pH and Paco₂ indicates that the primary process is metabolic acidosis. Figure 2 summarizes the analysis of this case.^{12-14,16,17}

METABOLIC ACIDOSIS

The patient in Case 1 demonstrates metabolic acidosis, which is classified as anion gap metabolic acidosis or non-anion gap metabolic acidosis by comparing sodium, chloride, and HCO₃.^{4,12} (See <https://www.mdcalc.com/calc/1669/anion-gap>.)

Anion Gap Metabolic Acidosis

The first step in analyzing metabolic acidosis is calculating the anion gap^{12,13} (Figure 3^{2-5,18}). Anion gap metabolic acidosis is indicated by an elevated anion gap (greater than 12 mEq/L [12 mmol/L]). The etiologies of anion gap metabolic acidosis can be remembered using the mnemonic CATMUDPILES¹⁹ (Table 3^{3,5,12,19,20}). For anion gap metabolic acidosis, calculating the osmolal gap (measured – calculated osmolality; <https://www.mdcalc.com/calc/91/serum-osmolality-osmolality>) can help differentiate between exogenous (high osmolal gap) and endogenous (normal osmolal gap) etiologies.¹⁴ Common endogenous causes include lactic acidosis (from sepsis, shock or hypoperfusion of organs, or

hypoxia), ketoacidosis (from uncontrolled diabetes or severe malnutrition), and uremia or kidney failure. Exogenous etiologies can be remembered using the mnemonic MADGAS²⁰ (Table 3^{3,5,12,19,20}).

Non-Anion Gap Metabolic Acidosis

Non-anion gap metabolic acidosis is diagnosed if the anion gap is not elevated. Etiologies can be remembered using the mnemonic HARDUP¹² (Table 3^{3,5,12,19,20}). Non-anion gap metabolic acidosis can result from kidney or gastrointestinal etiologies or infusion of large volumes of intravenous fluids

TABLE 2	
Compensation Rules	
Primary acid-base disorder	Expected compensation
Metabolic acidosis	Paco ₂ = HCO ₃ + 15
Metabolic alkalosis	Paco ₂ = 40 + [0.7 × (HCO ₃ – 24)]
Respiratory acidosis	HCO ₃ = 24 + [1 × (Paco ₂ – 40)/10] (acute) HCO ₃ = 24 + [3.5 × (Paco ₂ – 40)/10] (chronic)
Respiratory alkalosis	HCO ₃ = 24 – [2 × (40 – Paco ₂)/10] (acute) HCO ₃ = 24 – [5 × (40 – Paco ₂)/10] (chronic)
HCO ₃ = bicarbonate.	
Information from references 3 and 5.	

containing chloride (eg, normal saline) during resuscitation of critically ill patients.²¹⁻²⁴

If the cause of non-anion gap metabolic acidosis is unclear, then calculating the urine anion gap helps differentiate between renal and nonrenal etiologies.^{13,25} A normal urine anion gap indicates appropriate renal ammonia excretion and a nonrenal underlying cause (eg, diarrhea, total parenteral nutrition, high-volume intravenous fluids containing chloride). An abnormal urine anion gap suggests impaired renal ammonia excretion, such as in renal tubular acidosis.²⁶⁻²⁸

Further analysis of renal tubular acidosis is complicated, and consultation with an intensive care specialist or nephrologist may be beneficial. In general, treatment of renal tubular acidosis relies on reversal of the underlying kidney disease (eg, antibiotic use for kidney infection, improvement of renal perfusion from shock or sepsis, or removal of offending drugs or toxins).¹¹

Gap-Gap Analysis

The gap-gap ($\Delta\Delta$) analysis is the final step in metabolic acidosis assessment. A significant difference (exceeding 6 mEq/L) between the change in serum HCO_3^- ($\Delta\text{HCO}_3^- = 24 - \text{measured } \text{HCO}_3^-$) and change in anion gap ($\Delta\text{AG} = \text{calculated anion gap} - 12$) suggests an additional acid-base disturbance, as is seen in an anion gap metabolic acidosis (eg, diabetic ketoacidosis) complicated by a metabolic alkalosis (eg, contraction alkalosis due to vomiting).^{16,17,19} Treatment of metabolic acidosis with sodium HCO_3^- did not improve outcomes in multiple recent studies and may have negative consequences, such as paradoxical intracellular acidosis, hypocalcemia, or hypernatremia.³

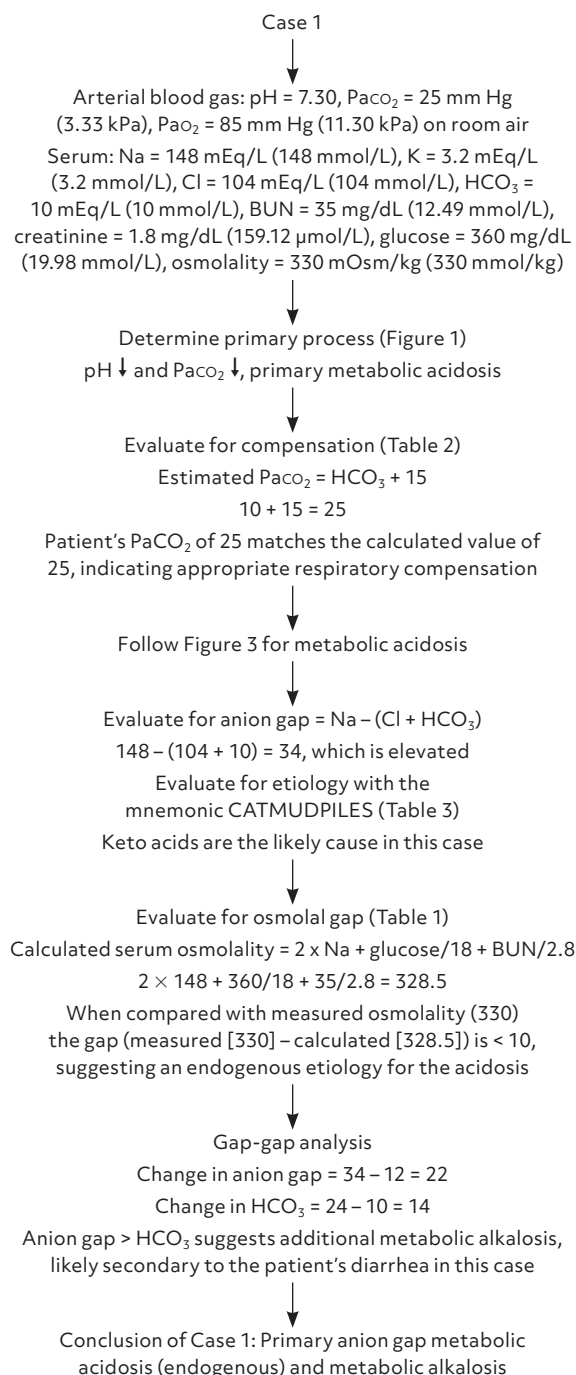
In Case 1, the patient demonstrated anion gap metabolic acidosis, likely due to diabetic ketoacidosis and acute kidney injury. Respiratory compensation was appropriate. However gap-gap analysis revealed metabolic alkalosis, likely due to several days of preceding diarrhea. Treatment focused on addressing the primary issues: intravenous hydration to correct fluid deficits and reinitiation of insulin to manage diabetic ketoacidosis. With these interventions, the patient's acid-base status, kidney function, and mental status improved rapidly.

CASE 2

A 56-year-old patient with a history of hypertension and chronic obstructive pulmonary disease was admitted from the emergency department with a 1-week history of nausea, vomiting, and diarrhea. On examination, the patient was afebrile, blood pressure was 98/65 mm Hg, heart rate was 118 beats per minute, and respiratory rate was 28 breaths per minute. Laboratory results were as follows:

- Arterial blood gas: pH = 7.52, Paco_2 = 45 mm Hg (5.99 kPa), PaO_2 = 82 mm Hg (10.91 kPa) on room air
- Serum: sodium = 145 mEq/L (145 mmol/L), potassium = 3.2 mEq/L, chloride = 98 mEq/L (98 mmol/L), HCO_3^- = 38 mEq/L (38 mmol/L), urea nitrogen = 15 mg/dL (5.35 mmol/L), creatinine = 1.2 mg/dL (106.08 $\mu\text{mol/L}$), glucose = 185 mg/dL (10.27 mmol/L)

FIGURE 2



BUN = serum urea nitrogen; Cl = chloride; HCO_3^- = bicarbonate; K = potassium; Na = sodium.

Case 1. Start with Figure 1 to determine the primary process (metabolic acidosis), then evaluate for compensation by referring to Table 2. Since this is a metabolic acidosis, follow the algorithm in Figure 3 to complete the analysis.

Information from references 12-14, 16, and 17.

- Spot urine chloride less than 10 mEq/L (10 mmol/L)

Comparison of the pH and P_{aCO_2} indicates that the primary process is metabolic alkalosis. Figure 4 summarizes the analysis for this case.^{29,30}

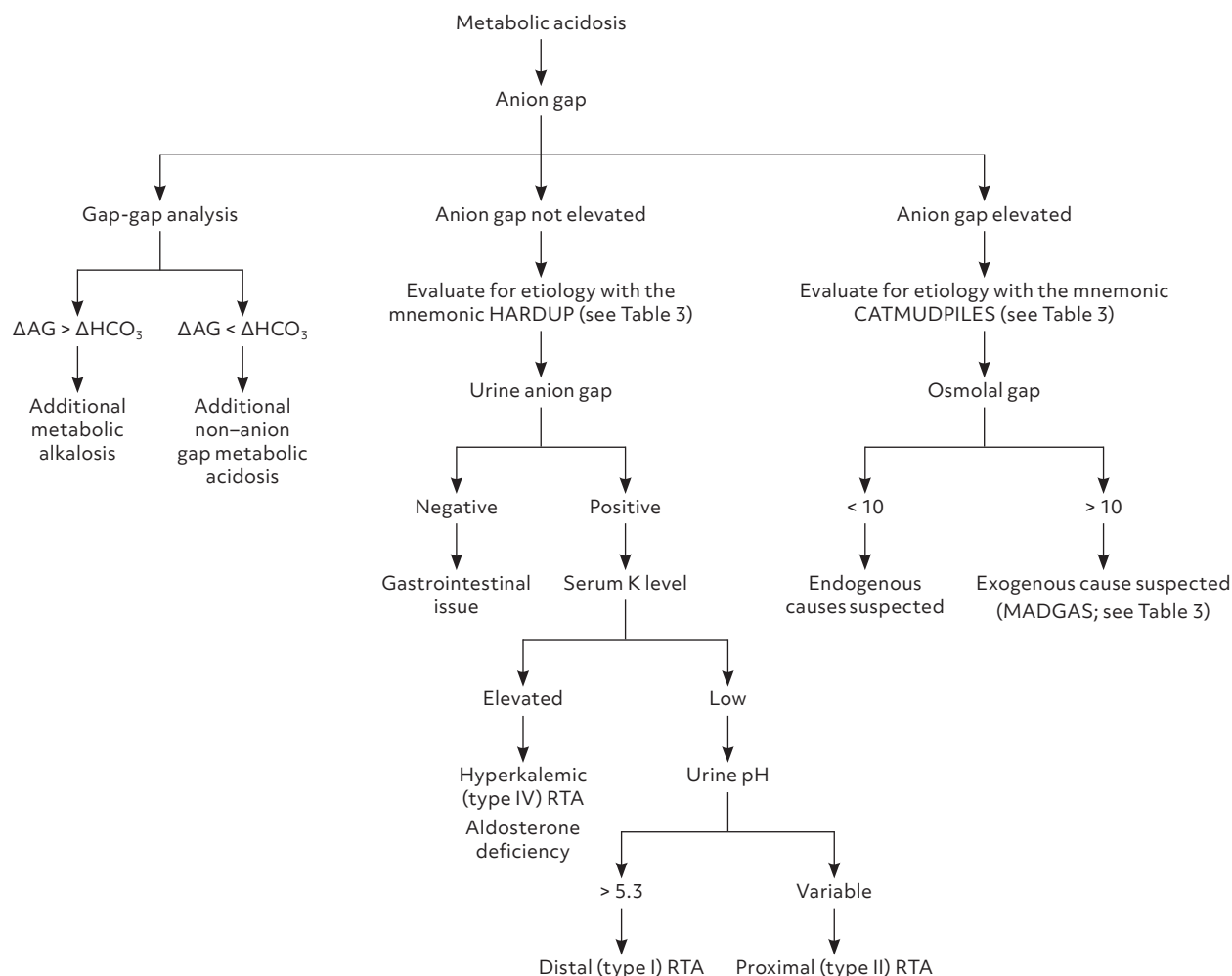
METABOLIC ALKALOSIS

Metabolic alkalosis is classified as chloride depletion or non-chloride depletion metabolic alkalosis.^{29,30} Chloride depletion alkalosis, often due to vomiting, diarrhea, or chronic diuretic use, is characterized by low urine chloride levels (20 mEq/L [20 mmol/L] or less) and typically improves with saline infusion.

For non-chloride depletion alkalosis (urine chloride level greater than 20 mEq/L), the next step is to evaluate volume status and blood pressure.^{11,18} Elevated blood pressure and volume status suggest hypermineralocorticoidism, warranting serum renin and aldosterone testing to guide further evaluation^{31,32} (Figure 5^{18,29,30,32}). Normal volume status and blood pressure may indicate primary kidney disorders such as Bartter syndrome or Gitelman syndrome.³² Fluctuating levels can be due to diuretic use, often needed in the care of critically ill patients with volume overload, or fluid resuscitation.^{11,18,30,32}

Metabolic alkalosis can also occur when overcompensating for an initial respiratory acidosis, particularly in patients with

FIGURE 3



AG = anion gap; HCO_3^- = bicarbonate; K = potassium; RTA = renal tubular acidosis.

Metabolic acidosis. The evaluation starts with calculating anion gap and, if needed, osmolal gap. For patients with a primary metabolic acidosis, a gap-gap analysis can help determine if an additional acid-base disturbance exists. If the anion gap is not elevated, the urine anion gap is calculated to differentiate etiologies. Serum potassium level and urine pH help differentiate various types of renal tubular acidosis.

Information from references 2-5 and 18.

chronic obstructive lung disease with chronic hypercapnia. Such patients compensate for the respiratory acidosis by gradually increasing their HCO_3^- level. Overzealous respiratory correction through mechanical ventilation (eg, bilevel positive airway pressure) can lead to a rapid decrease in PaCO_2 whereas HCO_3^- levels decrease gradually, resulting in metabolic alkalosis.^{15,32}

Case 2 showed primary chloride depletion metabolic alkalosis due to gastrointestinal loss of chloride with persistent

vomiting and diarrhea. Metabolic alkalosis resolved the next day after intravenous hydration was initiated. Respiratory alkalosis also resolved as the patient's volume depletion resolved.

RESPIRATORY ACIDOSIS AND ALKALOSIS

Respiratory acidosis results from hypoventilation, leading to increased PaCO_2 . The most common cause is chronic lung disease. An increasingly encountered cause is permissive hypercapnia, used in the treatment of acute lung injury or acute respiratory distress syndrome. Respiratory alkalosis results from hyperventilation, often seen in critically ill patients with sepsis, chronic liver disease, or inadequate sedation or pain control.¹⁵

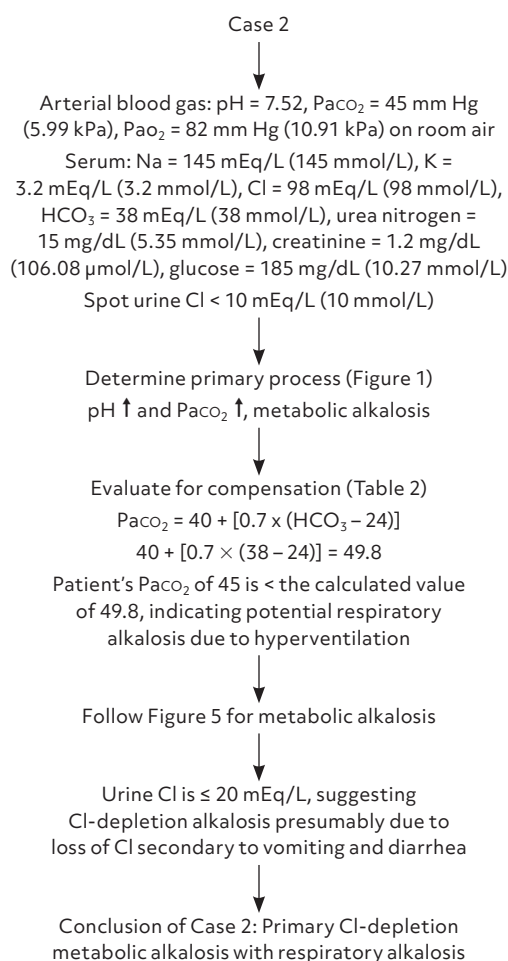
TABLE 3

Mnemonics for Acidosis Causes

Acid-base conditions	Mnemonic	Causes
Anion gap metabolic acidosis	CATMUDPILES	Carbon monoxide Alcohol, alcoholic ketoacidosis Toluene (glue sniffing) Methanol Uremia Diabetic ketoacidosis Paraldehyde, Isoniazid, iron, isopropyl alcohol Lactic acidosis Ethylene glycol (antifreeze) Salicylates
Exogenous high osmolal gap	MADGAS	Mannitol Alcohols Diatrizoate (imaging contrast) Glycerol Acetone Sorbitol
Non-anion gap metabolic acidosis	HARDUP	Hyperalimentation Adrenal insufficiency, acetazolamide Renal tubular acidosis Diarrhea and diuretics Ureteroenteric fistula Pancreaticoenteric fistula

Information from references 3, 5, 12, 19, and 20.

FIGURE 4

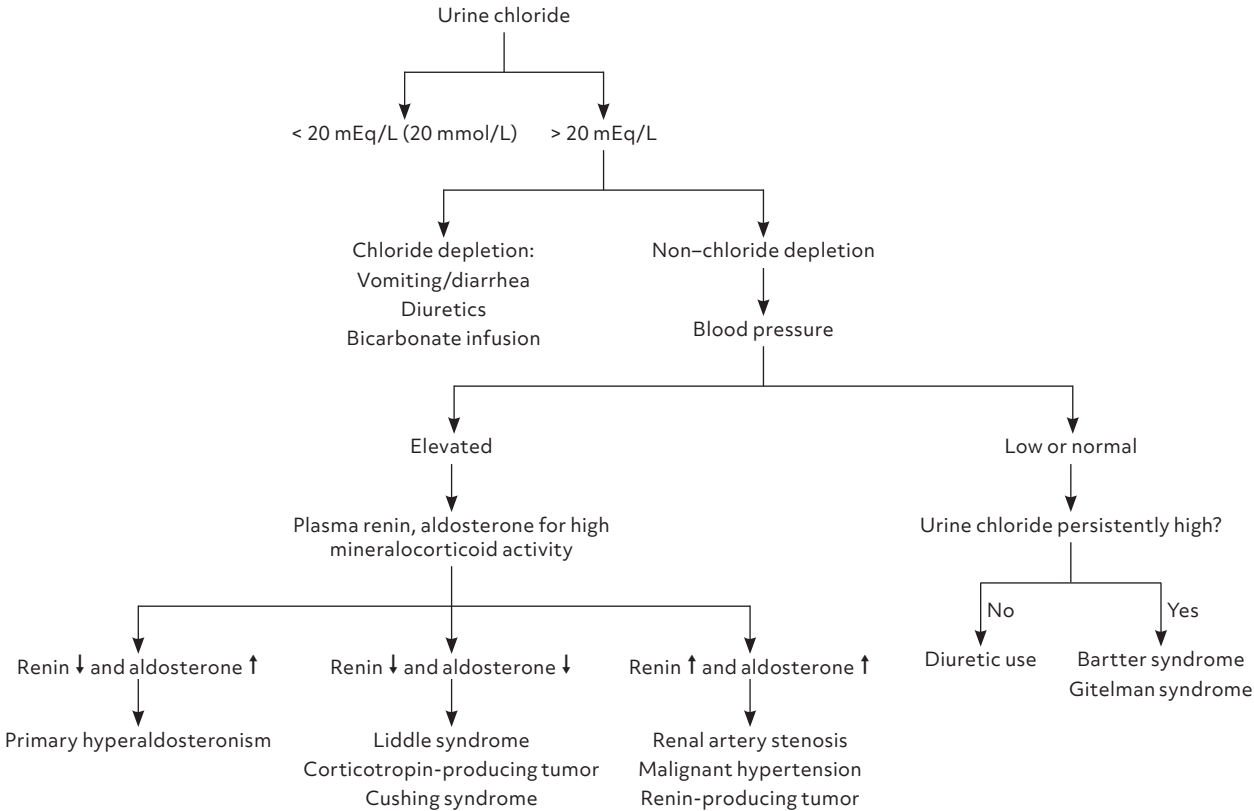


Cl = chloride; HCO_3^- = bicarbonate; K = potassium; Na = sodium.

Case 2. Start with Figure 1 to determine the primary process, then evaluate for compensation by referring to Table 2. Since this is a metabolic alkalosis, follow the algorithm in Figure 5 to complete the analysis.

Information from references 29 and 30.

FIGURE 5



Metabolic alkalosis. Urine chloride measurement is useful to determine if metabolic alkalosis is a chloride depletion alkalosis or non-chloride depletion alkalosis. If non-chloride depletion, volume status and blood pressure should be examined. Renin and aldosterone levels can help differentiate between etiologies in non-chloride depletion alkalosis associated with elevated blood pressure.

Information from references 18, 29, 30, and 32.

If the primary process is respiratory, the patient should be quickly evaluated for additional acid-base disorders. In acute settings, this is estimated by checking the pH change associated with PaCO_2 change: a 10-mm Hg change of PaCO_2 typically correlates with a 0.08 change in pH ($\Delta 0.08$ pH correlates to $\Delta 10$ mm Hg PaCO_2).¹⁵ If the pH change exceeds this ratio, additional metabolic derangement should be suspected and investigated using the metabolic acidosis or alkalosis algorithms. Additional metabolic derangement should also be considered when the compensation in HCO_3^- is greater than or less than expected for an acute or chronic respiratory process² (Table 2^{3,5}).

Data Sources: We conducted a PubMed search using the key terms acid-base disorders, anion gap, and blood gas analysis and including randomized controlled trials, clinical trials, reviews, and some case series. We focused on clinically relevant articles, and we expanded our search for recommendations and guide-

lines, including DynaMed, Cochrane, and ACCESS. We found only a limited number of articles came up under our search that were clinically relevant. Search dates: December 20, 2023, and November 21, 2024.

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